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Case Report

Brunner's gland hyperplasia: a rare case report

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ABSTRACT

A rare case of Brunner's gland hyperplasia mimicking duodenal malignancy is reported. A 21-year old man had a lesion in the second portion of the duodenum on endoscopy. The biopsy specimen suggested moderate and chronically active duodenitis. However, since the CT features strongly suggested malignancy, pyloroduodenotomy, excision of the lesion and Heineke-Mikulicz pyloroplasty was performed. The final pathological diagnosis was Brunner's gland hyperplasia.

Keywords: Brunner's gland, Duodenum, Heineke-Mikulicz pyloroplasty, Hyperplasia

INTRODUCTION

Brunner's gland, which was accurately described by Brunner in 1688, is a gland in the submucosa of the duodenum, which has a main physiological function of secreting an alkaline-based mucus to protect the duodenal lining from the acid secreted in the stomach.[1] Brunner's gland hyperplasia also known as Brunneroma or polypoid hamartoma is a rare, benign, proliferative lesion arising from the Brunner's glands of the duodenum. It accounts for 10.6% of benign tumors of the duodenum and rarely presents with any symptoms.[2] The first case of Brunner's gland hyperplasia was reported in 1835 in a patient with fatal duodenal intussusception.[3] Patients with Brunner's gland hyperplasia are usually asymptomatic, and the lesions are discovered incidentally. However, Brunner's gland hyperplasia can lead to significant symptoms including gastrointestinal bleeding, abdominal pain and intestinal obstruction. We report a rare case of Brunner's gland hyperplasia mimicking duodenal malignancy.

CASE REPORT

A 21 years old man presented with epigastric discomfort, weight loss, nausea and vomiting for 3 months duration. Total leucocyte count was 10,950cells/cumm (normal range 4000-10000cells/cumm), other laboratory values were within normal limits. A 3-D abdominal computer tomography (CT) was then performed, which showed a 2.5cm-sized mass in the second portion of duodenum with loss of fat plane between the duodenal mass and pancreas, which gave suspicion of a pancreatic invasion and a 7mm-sized sub pyloric node.

The stage based on the CT finding was stage III (T3, N1, Mx). An upper GI endoscopy was performed, which revealed an infiltrating type of mass at the second portion of the duodenum with luminal narrowing, so the scope could not pass through the narrowed lumen (Figure 1). Endoscopic biopsy revealed moderate and chronically active duodenitis.

Since the patient had symptoms of recurrent vomiting and weight loss due to duodenal obstruction and the finding from the abdominal CT was suspicious of duodenal cancer in the second portion with a pancreatic invasion, patient was planned for exploratory laparotomy, kocherisation was done and a small lesion approximately 1.5 to 2cm was felt in the second part of the duodenum

without any involvement of the seromuscular layers and adjacent pancreas.

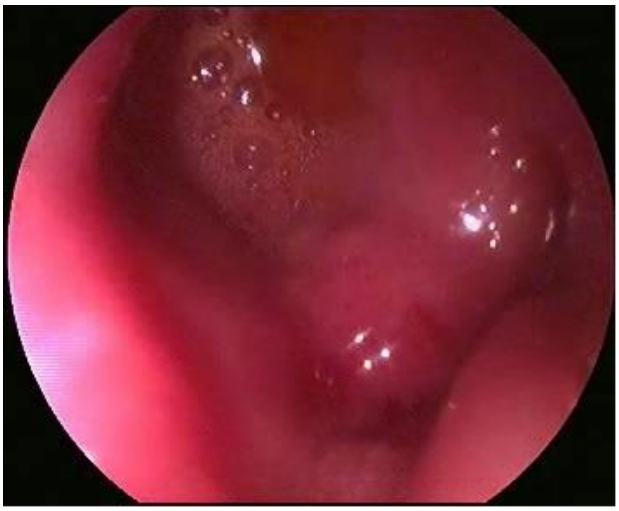

Figure 1: Endoscopic picture showing mass at second portion of duodenum with luminal narrowing.

Pyloroduodenotomy was done, A well circumscribed lesion of approximately 1.5cm found in the second part of duodenum. It was excised and Heineke- Mikulicz was done, after confirmation of frozen section biopsy report showing pancreatic heterotopia without evidence of malignancy. The final histopathological report showed a diffuse nodular type of Brunner's glands hyperplasia with inflammation of the ampulla, *H. pylori* organisms was seen and there was reactive lymphadenopathy of sub pyloric node (Figure 2).

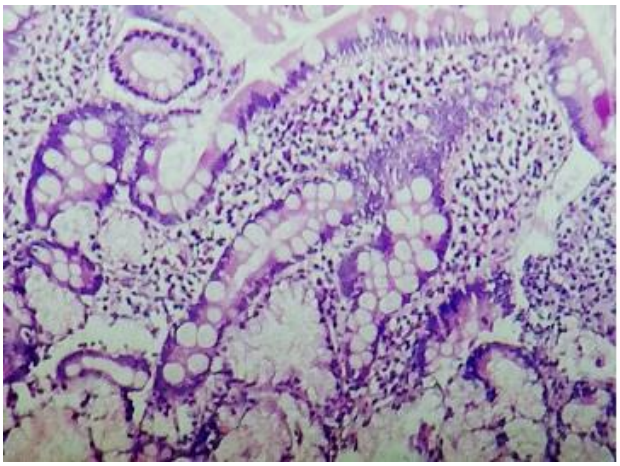

Figure 2: HPE showing diffuse nodular type of brunner's gland hyperplasia.

Pancreatic heterotopia was found in the subserosa of the duodenum. Finally, the patient was diagnosed with a diffuse nodular type of Brunner's gland hyperplasia with duodenal obstruction. The postoperative period was uneventful, patient was treated with *H. pylori* kit for 2 weeks. Repeat upper GI endoscopy was done after 2 months and found to be normal. The patient has been on a regular follow up till date and has shown no clinical signs of recurrence.

DISCUSSION

The cause of Brunner's gland hyperplasia is thought to be chronic inflammation, chronic stimulus by excessive secretion of gastric acids, or a decrease in pancreatic exocrine function. However, it has recently been thought that the hyperactivity of the exocrine modulating factors (hormone, vagus nerve and intestinal mucous membrane factor) are the major causes of Brunner's gland hyperplasia.[4] It is distributed mainly on the duodenal bulb (57%), second portion of the duodenum (27%), and its third portion (7%). It is rarely seen on the pylorus (5%), jejunum (2%), or the terminal ileum (2%).

The clinical manifestations of Brunner's gland hyperplasia are nonspecific, such as epigastric discomfort, abdominal distension, or dyspepsia etc. Occasionally, it can lead to hemorrhage, acceleration of peristalsis, diarrhoea, transient and partial bowel obstruction and duodenal intussusception due to the mass.

In 1934, Feyrter classified the abnormal glandular proliferation in to the following three types: diffuse hyperplasia (type 1), nodular hyperplasia (type 2), and adenomatous hyperplasia (type 3).[5-7] This nodular hyperplasia (type 2) is often mistaken for a malignancy. The diagnosis of Brunner's gland hyperplasia is supported by endoscopy and radiological findings. However, the sensitivity of endoscopy is 72-89% and occasionally, endoscopy is not useful in making the diagnosis.ss[5-7] Thus, we found that abdominal CT was useful in this case.

Endoscopic biopsy usually gives a negative result, because the tumour is almost entirely covered with thick intact duodenal mucosa and the biopsy is often not deep enough to reach the submucosal tumor tissue.[8] Since in the present case an endoscopic biopsy from the tumor did not show any histological features of malignancy, and CT features suggested the possibility of malignancy, surgery was planned, even though the frozen biopsy showed no evidence of malignancy, we decided to carry on with exploratory laparotomy and a circumscribed lesion of approximately 1.5cms found in the second part of duodenum, it was excised Heineke-Mickulicz pyloroplasty was done.

Another unique aspect of this case is the coexistence of Brunner's gland hyperplasia and pancreatic heterotophia. The relation of ectopic pancreas and Brunner's gland hyperplasia is not clear. Although there is less connection between the cause of Brunner's gland hyperplasia and the pancreatic heterotophia, we need further studies about the relationship of Brunner's gland hyperplasia with pancreatic heterotophia.

It is still controversial whether asymptomatic Brunner's gland hyperplasia or adenoma found incidentally needs surgical removal. Some think that it needs no treatment, whereas others believe that they should undergo endoscopic excision in order to prevent complications. There have been several reports that,[9,10] Brunner's gland hyperplasia or adenoma could give rise to acute profuse bleeding which results in shock. It has also been reported that a pedunculated polyp with carcinoma is thought to have developed due to induction of gastric-foveolar differentiation in a manner very similar to that of gastric metaplasia in hyperplastic Brunner's glands.[11]

CONCLUSION

In conclusion, although Brunner's gland hyperplasia mimicking duodenal tumor is very rare, it should be taken in to consideration in the differential diagnosis of duodenal tumor.

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